Severe apnea and bradycardia in a term infant

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Abstract: Case report : This male infant was born at 39 4/7 weeks of gestation to a 24-year-old G1/P1 following an uncomplicated pregnancy. During labor, when signs of fetal distress were noted on the CTG and the amniotic fluid became meconium-stained, it was decided to accelerate delivery by vacuum extraction. Apgar scores were 7, 8, and 10 after 1, 5, and 10 minutes, respectively, and the arterial cord blood pH was 7.34. Physical examination was unremarkable apart from a parieto-occipital vacuum extraction mark. Birth weight was 3060 g, head circumference 34 cm and length 50 cm (all 10th to 25th percentile). The infant was brought to the maternity ward with his mother.

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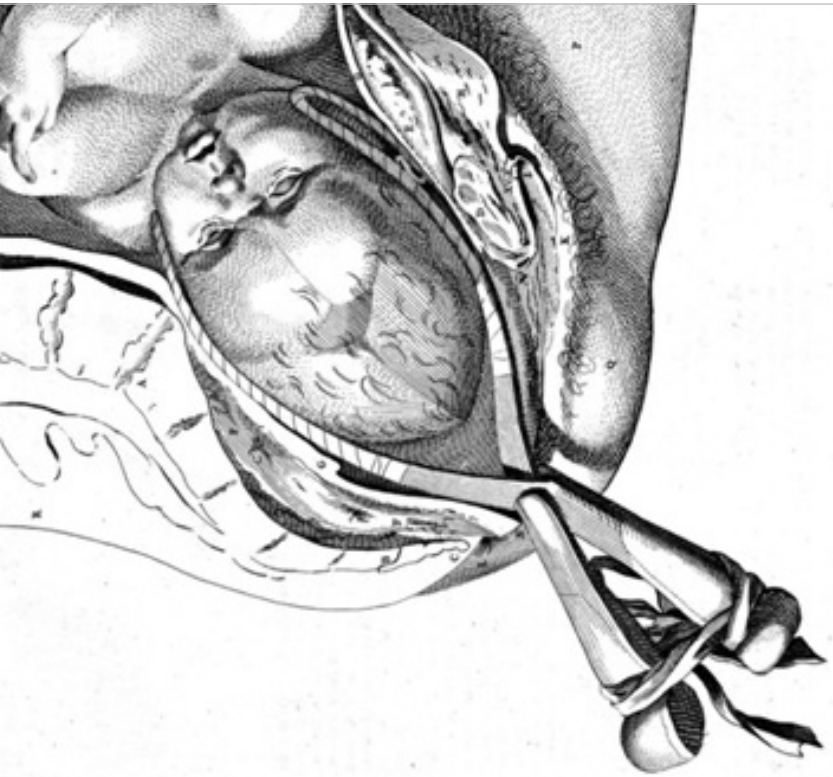

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This male infant was born at 39 4/7 weeks of gestation to a 24-year-old G1/P1 following an uncomplicated pregnancy. During labor, when signs of fetal distress were noted on the CTG and the amniotic fluid became meconium-stained, it was decided to accelerate delivery by vacuum extraction. Apgar scores were 7, 8, and 10 after 1, 5, and 10 minutes, respectively, and the arterial cord blood pH was 7.34. Physical examination was unremarkable apart from a parieto-occipital vacuum extraction mark. Birth weight was 3060 g, head circumference 34 cm and length 50 cm (all 10th to 25th percentile). The infant was brought to the maternity ward with his mother.

At the age of 14 hours, the boy was noted to be pale, floppy, and hypothermic (36.0 °C). He was admitted to the neonatal intensive care unit, where a bulging fontanelle as well as intermittent severe apnea and bradycardia became apparent. Consequently, he was intubated and ventilated. Neither seizures nor other signs of elevated intracranial pressure were detected.

Blood gas analysis on admission revealed uncompensated metabolic acidosis (pH 7.24, pCO_2 6.1 kPa, BE -7.8 mmol/l). Suspecting early-onset neonatal infection, empiric antibiotic therapy (amoxicillin, gentamicin) was initiated, and stopped 48 hours later when blood and cerebrospinal fluid cultures remained negative. Mild anemia was found (hematocrit 38 %), and a single packed red blood cell transfusion was given.

Platelet and reticulocyte counts as well as a coagulation profile were normal.

Cranial ultrasonography showed dilation of the lateral and third ventricles. Moreover, a round, echo-dense structure within the cerebellar vermis was suggestive of bleeding within the cerebellum (Fig. 1, 2). Emergency CT scan confirmed the diagnosis of cerebellar hemorrhage and hydrocephalus (Fig. 3). Despite minimal ventilator settings, extubation failed because of apnea.

The infant was transferred to our department of pediatric surgery, where the hemorrhage was shown to be progressive on follow-up CT scan. The cause of the infratentorial hemorrhage remained unclear. Craniotomy and hematoma evacuation were performed and an external cerebrospinal fluid drain was inserted. The postoperative course was uncomplicated (Fig. 4). The drain was removed on the second postoperative day. The boy was discharged home on the 14th day of life. At the age of 2 months, muscular hypotonia of the trunk and diminished head control were noted. The most recent follow-up examination at the age of 6 months revealed no neurological deficits except for mild increase of muscle tone.

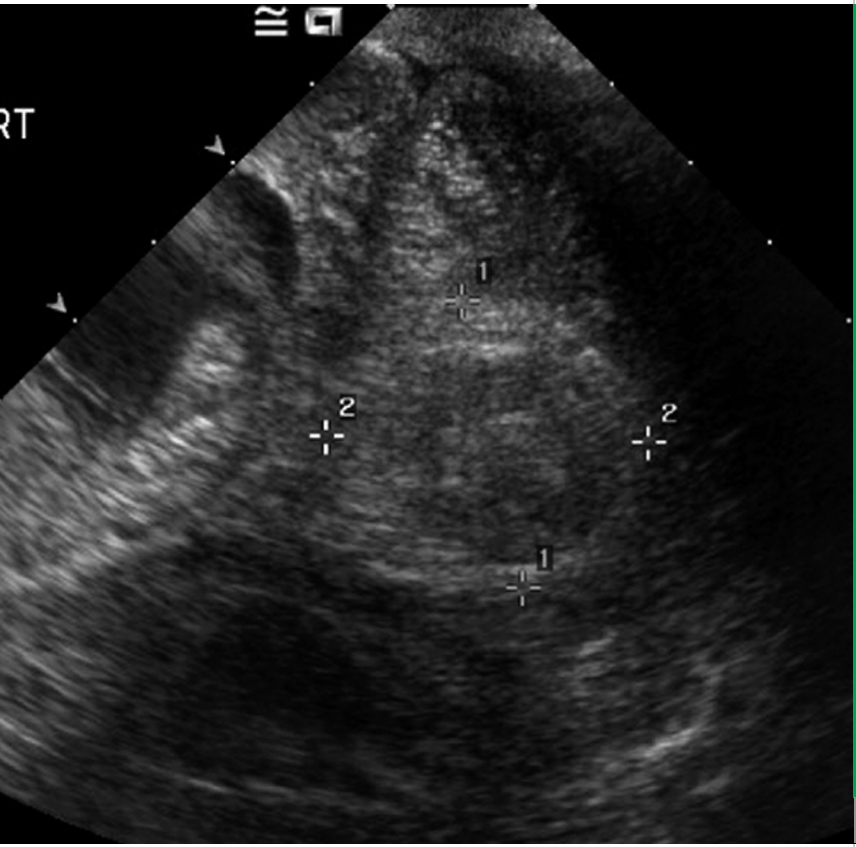

Fig. 1

Cerebral ultrasound (second day of life): midline lesion in the cerebellar region.

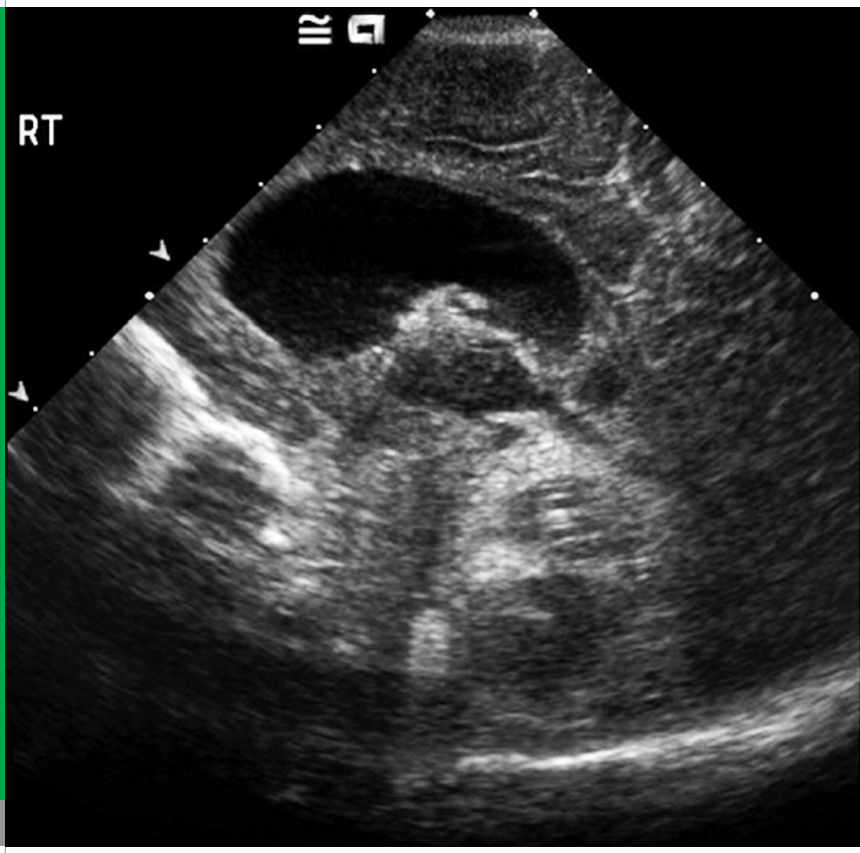

Fig. 2

Cerebral ultrasound (second day of life): ventricular dilatation.

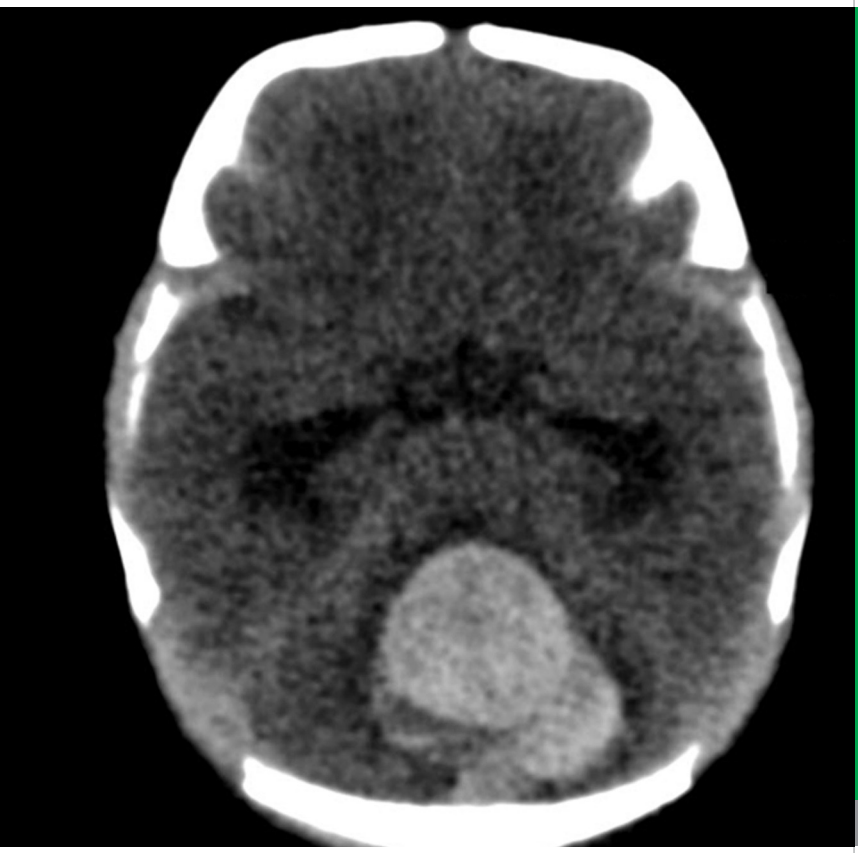

Fig. 3

CT scan on the third day of life: cerebellar hemorrhage.

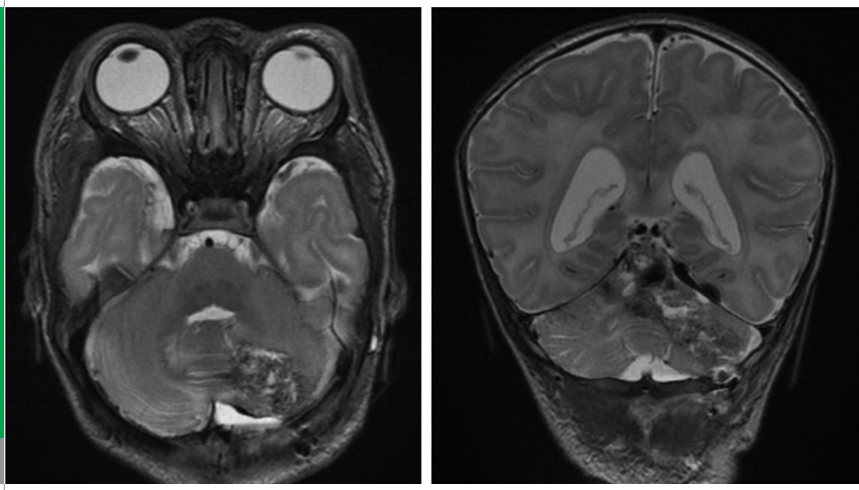

Fig.4

Postoperative MRI on the sixth day of life: residual changes following evacuation of the infratentorial hemorrhage.

Compared with premature infants, intracranial hemorrhage in term infants is uncommon and differs in location, etiology, presentation, and neurological outcome (1). The incidence of intracranial hemorrhage in term infants is not known and difficult to determine due to a substantial number of asymptomatic cases. A retrospective study of 65 asymptomatic term infants born via vaginal delivery and undergoing cranial MRI between the ages of one and five weeks showed a prevalence of 26 % (2), suggesting that asymptomatic intracranial hemorrhage is much more frequent than previously thought. In this study, the majority (67 %) of intracranial hemorrhages was subdural, and most were infratentorial, but there were no cerebellar hemorrhages (2). In contrast, Miall et al. (3) analyzed retrospectively all neonatal MR brain scans obtained at the Leeds General Infirmary over a five-year-period. This large cohort of 558 newborns comprised infants admitted to the intensive care unit with abnormalities on cranial ultrasonography, abnormal neurological examination that required further neuroimaging as well as normal infants who had been scanned for research purposes. Out of these newborns, 20 (3.6 %, 10 term, 10 preterm) had abnormalities in the posterior fossa. Nine (1.6 %) had cerebellar hemorrhages, of which only one was a term infant (3).

Cerebellar hemorrhage in term infants is frequently associated with traumatic delivery (4). Prenatal and intrapartum risk factors include primiparity, advanced

maternal age, abnormal fetal heart rate, instrumental delivery, and emergency Cesarean section (4); three of these factors were present in our case. More unusual causes are primary clotting abnormalities or congenital vascular abnormalities (5). Cerebellar hemorrhagic injury following traumatic delivery is believed to result from severe distortion and disruption of the venous structures within the skull, leading to laceration of the tentorium or falx, or due to direct traumatic cerebellar laceration along the vermis (4). However, in a study by Towner et al. (6), the incidence of intracranial hemorrhages in infants born by Cesarean section during labor was equal to that in infants born by vacuum extraction, and the frequency of intracranial hemorrhages in infants born by caesarean section with no labor did not differ significantly from that in infants delivered spontaneously. Towner et al. concluded that a substantial proportion of the morbidity associated with operative vaginal delivery might be due to an underlying abnormality of labor rather than to the delivery procedure (6).

Two thirds of infants with cerebellar injury develop apneic episodes and nearly one third develop clinical seizures (4). Bradycardia and fall in hematocrit are also characteristic (7). The neurodevelopmental outcome is variable, ranging from normal to significant and wide-ranging disabilities, predominantly gross motor and expressive language deficits and behavioral problems (4).

CONCLUSION

Cerebellar hemorrhage in term newborns may be asymptomatic or present with signs of elevated intracranial pressure, seizures or symptoms mimicking neonatal infection. In term neonates with apnea and bradycardia, intracranial hemorrhage is an important differential diagnosis to consider. There is a strong association - but no causality - between traumatic delivery, especially vacuum extraction, and intracranial hemorrhage. Apart from instrumental delivery, risk factors for cerebellar injury include primiparity, advanced maternal age and abnormal fetal heart rate.

REFERENCES

1. Gupta SN, Kechli AM, Kanamalla US. Intracranial hemorrhage in term newborns: management and outcomes. Pediatr Neurol 2009;40:1-12

2. Looney CB, Smith JK, Merck LH, et al. Intracranial hemorrhage in asymptomatic neonates: Prevalence on MR images and relationship to obstetric and neonatal risk factors. Radiology 2007;242:535-41

3. Miall LS, Cornette LG, Tanner SF, Arthur RJ, Levene MI: Posterior fossa abnormalities seen on magnetic resonance brain imaging in a cohort of newborn infants. J Perinatol 2003; 23:396-403

4. Limperopoulos C, Robertson RL, Sullivan NR, Bassan H, du Plessis AJ. Cerebellar injury in term infants: clinical characteristics, magnetic resonance imaging findings, and outcome. Pediatr Neurol 2009;41:1-8

5. Rutherford MA. MRI of the neonatal brain. Part 4, chapter 9, Ebook, ISBN 0 7020 2534 8, available from *www.mrineonatalbrain.com*

6. Towner D, Castro MA, Eby-Wilkens E, Gilbert WM. Effect of mode of delivery in nulliparous women on neonatal intracranial injury. N Engl J Med 1999;341:1709-1714

7. Shankaran S. Intracranial hemorrhage in newborn infants. Indian J Pediatr 1983;50:353-362

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